Preterm infant with Clostridium perfringens intestinal gangrene

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SWISS SOCIETY OF NEONATOLOGY

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September 2018

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Title figure:
(source: cdn.bionews-tx.com)

This premature baby was delivered at 25 0/7 weeks of gestation with a birth weight of 840 g by Cesarean section due to chorioamnionitis. Apgar scores were 4, 4 and 5 at 1, 5 and 10 minutes, respectively. Surfactant was administered after intubation within the first minutes of life. Antibiotics were given for 48 hours; blood cultures and sepsis workup remained negative. Enteral feeding with formula milk was started on the first day of life (DOL) and increased daily reaching full feeds on DOL 8.

On DOL 12, the baby presented with progressive apneas and a distended abdomen. Therapy with amoxicillin/clavulanic acid and gentamicin was started and oral feedings were stopped. Abdominal X-rays showed advanced signs of necrotizing enterocolitis (NEC) with portal venous gas and pneumoperitoneum (Fig. 1, 2).

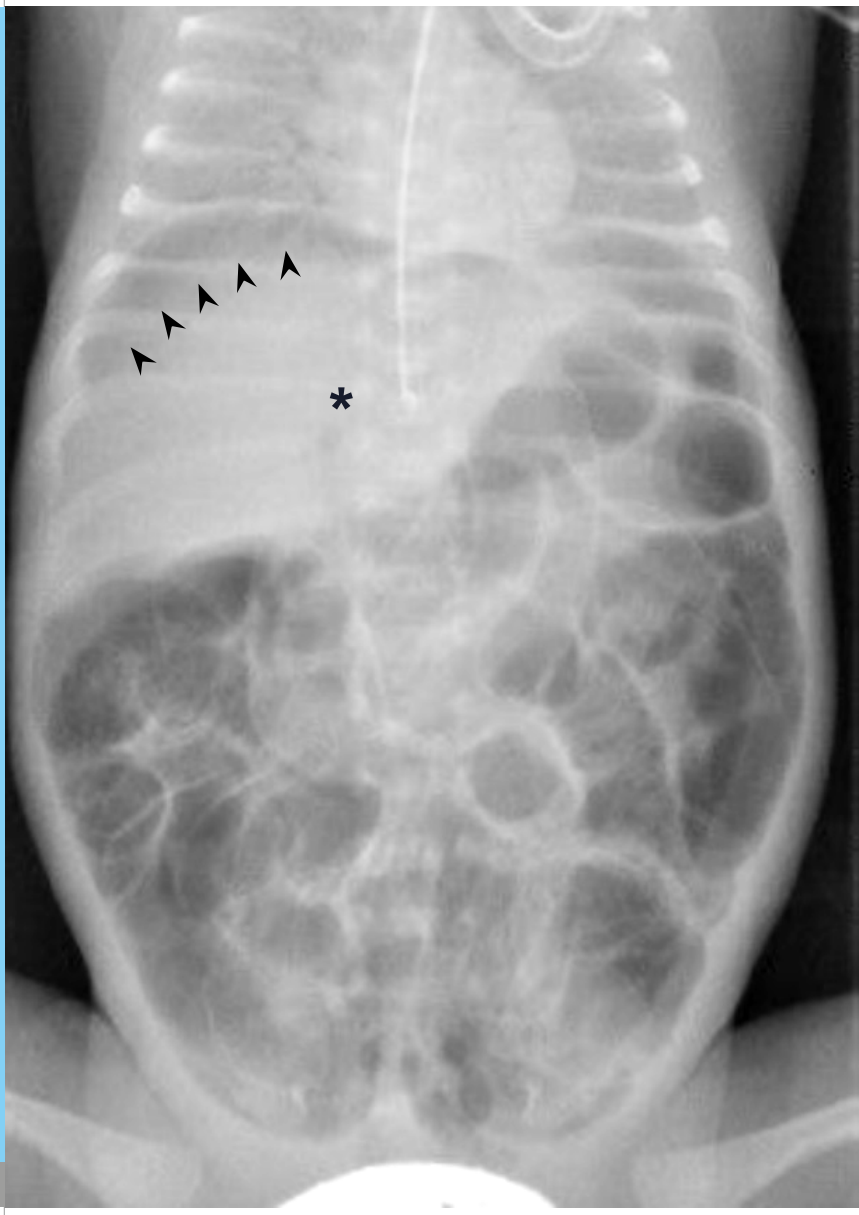

Fig. 1

Abdominal X-ray (ap view): dilated loops of bowel, intestinal pneumatosis, pneumoperitoneum (arrow heads) and portal venous gas (asterisk).

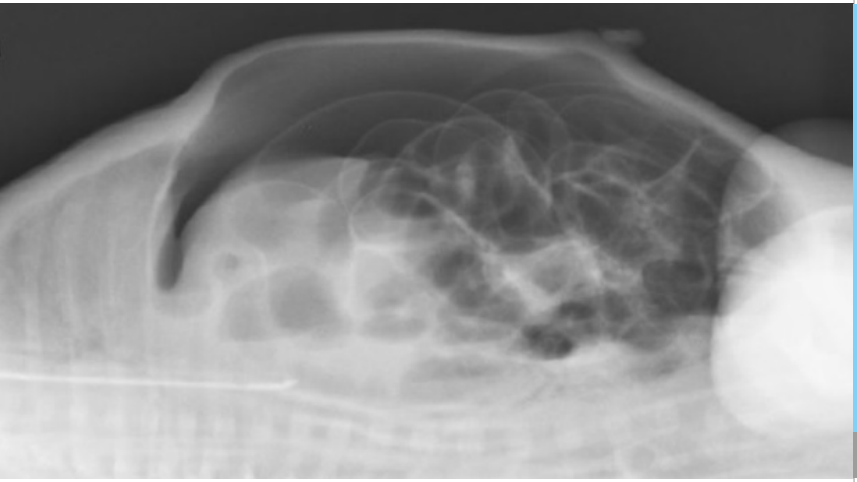

Fig. 2

Abdominal X-ray (lateral view, horizontal beam technique): pneumoperitoneum.

A peritoneal drain was inserted, and metronidazole was added. However, the baby deteriorated further and developed arterial hypotension with poor microcirculation, as well as coagulopathy. At this time, the abdomen was severely distended and discolored. Emergency laparotomy showed complete gangrenous necrosis of the small intestines as well as a partially liquified colon (Fig. 3).

Resection of the intestinal tract was not an option, and, with parental consent, palliative care was provided. The baby died less than 24 hours after the onset of the first symptoms (Table 1). Cultures from the peritoneal cavity showed Clostridium perfringens, whereas the blood culture and C-reactive Protein remained negative throughout the course.

Time point 0	0 + 7 hours	0 + 10 hours	0 + 18 hours	0 + 23 hours
Onset of first symptoms: • progressive apneas • slightly distended abdomen	**Clinical deterioration:** • portal venous gas • peritoneal drain inserted	**Further progression:** • severely distended and discolored abdomen • arterial hypotension	**Laparotomy:** • necrosis of small and large bowel	**Redirection of care:** • infant died one hour after extubation

Chronology of the clinical course.

Fig. 3

Intraoperative findings: extensive bowel necrosis consistent with gas gangrene.

DISCUSSION

NEC is still one of the leading causes of mortality in preterm infants. Despite having first been described more than fifty years ago, the incidence remains unchanged affecting approximately 7 % of preterm infants with birth weights less than 1500 g and up to 35 % of extremely low birth weight infants (1). Survivors may suffer from lifelong gastrointestinal dysfunction and neurological impairment (2).

NEC is characterized by acute bowel inflammation of different extent and localization, typically involving the ileum and proximal colon (3, 4). The pathogenesis seems to be multifactorial but is still not fully understood. Gut mucosal immaturity, impaired intestinal blood flow with ischemia causing mucosal lesions and an imbalance of the bacterial gut flora with overgrowth of pathogenic bacteria seem to be important factors (5). Intra- and postpartum antibiotics may contribute to gut flora imbalance.

A large variety of bacteria, viruses and fungi can be associated with NEC but there is no specific infectious agent (6). There are some reports describing the isolation of Clostridium perfringens from infants with NEC, suggesting that this microorganism may also play a role in the etiology of the inflammation (5).

Clostridium perfringens is a gram-positive anaerobic bacterium that can also be present in the gut of healthy children, particularly in hospitalized newborns (7).

Colonization with Clostridium perfringens is found in less than 10 % of preterm babies during the first days of life and increases up to 35 % in the first two weeks of life (8).

Bowel ischemia may trigger the conversion of clostridial spores into toxin-producing invading bacilli. The α-toxin is the most common toxin produced in human gas gangrene (5) and associated with massive gas production.

Some authors speculate that clostridial gas gangrene is a different entity than classical NEC observed in premature infants (1, 5). The severity of the clinical course with higher rates of morbidity and mortality in cases associated with Clostridium perfringens, as illustrated by the presented case, may support this hypothesis (1). The short interval between the first onset of symptoms and deterioration is presumably due to the short incubation time of the organism (6).

Our infant, in line with some other reports of preterm babies with intestinal gas gangrene, showed symptoms within the first two weeks of life, rapid clinical deterioration, massive pneumoperitoneum, intestinal pneumatosis, portal venous gas and signs of shock and coagulopathy. These complications seem to be more frequent in babies with clostridial intestinal gangrene compared to those with classical NEC (9).

Published cases with clostridial intestinal necrosis required surgery within 24 hours. Intraoperative findings demonstrated extensive gas gangrene and bowel resection was usually inevitable (9).

CONCLUSION

The presented preterm boy developed severe signs of enterocolitis on the 12th DOL. The clinical course, intraoperative findings and isolation of Clostridium perfringens indicate intestinal gas gangrene. It is still a matter of discussion whether intestinal gas gangrene should be considered a separate disease entity or an extreme form of NEC.

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SUPPORTED BY VIFOR PHARMA

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